"AN ANALYSIS OF SERUM ELECTROLYTE AND OSMOLALITY IN SURGICAL PATIENTS IN REFERENCE TO PERIOPERATIVE INFUSION"

THESIS

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1990 ONKAR NATH PANDEY

DEPARTMENT OF SURGERY
M.L.B. MEDICAL COLLEGE
JHANSI (U.P.)

C E R T I F I C A T E

This is to certify that the work entitled as " AN ANALYSIS OF SERUM ELECTROLYTE AND OSMOLALITY IN SURGICAL PATIENTS IN REFERENCE TO PERIOPERATIVE INFUSION" which is being submitted as THESIS for M.S.(General Surgery) examination, 1990 of Bundelkhand University, Jhansi has been carried out by DR. ONKAR NATH PANDEY, himself in this department.

He has put in the necessary stay in the department as required by the regulation of Bundelkhand University.

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This is to certify that DR. ONKAR NATH PANDEY has worked on " AN ANALYSIS OF SERUM ELECTROLYTE AND OSMOLALITY IN SURGICAL PATIENTS IN REFERENCE TO PERIOPERATIVE INFUSION" under my guidance and supervision.

His results and observations have been checked and verified by me from time to time.

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(ONKAR NATH PANDEY)

C O N T E N T S

INTRODUCTION

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Optimal management of the surgical patient today demands a thorough knowledge of the changes in fluid and electrolyte balance associated with the surgical procedure. With the advent of newer diagnostic methods a more acurate assessment of perioperative fluid and electrolyte alterations are now possible. The reasons of these changes are however, far less completely understood.

Acute changes in renal function in the operative and post operative period have been observed for many years and the decreased renal excretion of sodium in the post operative period is well documented since a long time. It has been recently established that the extracellular fluid volume, or more precisely the "functional extra cellular fluid volume is a major determinent of this renal sodium excretion.

It was demonstrated that the metabolic responses of the body to surgical stress profoundly alter the need for salt and water in the post operative period. As a result of these studies it had been customary for some years to limit the amounts of water and sodium administered in the post operative period. Most clinicians believed that such limitation reduces the incidence of

edema, dilutional hyponatraemia, and water intoxication, problems which had been noted to occur in the post operative patients. A number of recent reports have indicated that there is an acute deficit in the volume of extracellular fluid space during surgical trauma and that a large amount of fluid is needed to correct this deficit in the extracellular fluid volume. Also despite the fact that there is sodium retention after any kind of trauma, including surgery, in the early post operative period, there is still a tendency towards hyponatraemia probably because of an associated fluid retention. The isotonicity of body fluids is maintained between intracellular and extracellular fluid, further increases the hyponatraemia, which occurs after surgical procedure. The implication of this hyponatraemia, when seen in the light of type of fluid administration per operatively and post operatively and the effect of this fluid on the serum sodium level, is important for the clinician. In this context we needed to reexamine the usual concept of administerating sodium free fluid in the immediate post operative period.

AIMS OF STUDY

1. To document the sodium and potassium status pre operatively and to see the changes in the serum sodium and potassium in early post operative period.

2. To assess the effect of type of fluid given in the perioperative and early post operative period on the serum osmolality.

3. To assess the effect of type of fluid given in the perioperative and early post operative period on urinary sodium and urine volume.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

One of the most critical aspects of a surgical patient's care relates to the management of fluid and electrolytes, especially because the operative trauma imposes a great impact on the physiology of fluid and electrolytes within the body.

ANATOMY OF BODY FLUID

Total body water - Water constitutes 50-70% of total body weight. Moore et al (1963) have shown that total body water as a percentage of total body weight, decreases steadily and significantly with age to a low 52 and 47% in males and females respectively.

The water of the body is divided into three functional compartments. The fluid with in the body's diverse cell population, represents between 30 & 40% of the body weight. The extra cellular water represents approximately 20 percent of the body weight and is divided between the intravascular fluid, or plasma (5% of body weight) and the interstitial, or extravascular, extra cellular fluid (15% of body weight).

Intracellular fluid - The intracellular water is between 30 to 40 percent of the body weight. The chemical composi-

tion of the intra celluler fluid is as follows - Cations K^+ 150 Meq/Lt., Mg^{++} 40 Meq/Lt. Na^+ 10 Meq/Lt. Anion Po_4^{---} & So_4^{--} 150 Meq/Lt. HCo_3^- 10 Meq/Lt. Proteins 40 Meq/Lt.

The potassium and magnesium are the principal cations and phosphates & proteins the principal anion in intra celluler compartment.

Extra cellular fluid - The total extra celluler fluid volume represents 20 percent of the body weight. The extra celluler fluid compartment has two major subdivisions. The plasma volume is approximately 5% of the weight in the normal adult. The interstitial or extra vascular, extra celluler fluid volume is obtained by substracting the plasma volume from the measured total extra celluler fluid volume and constitutes approximately 15% of the body weight.

The interstitial fluid is further complicated by having, normally, a rapidly equilibrating or functional,component as well as several more slowly equilibrating, or relatively nonfunctioning, components. The non functioning components include connective tissue water as well as water that has been termed transcellular, which includes cerebrospinal and joint fluids. This non functional component normally represents only 10 percent of the interstitial fluid volume (1 - 2 percent) and is not to be confused with

" The osmolar concentration expressed as osmols per litre of solution". Osmoles is the number of osmotically active particles or ions per unit volume ".

The differences in the ionic composition of intra cellular and extra cellular fluid compartments are maintained by the cell wall, which functions as a semi-permiable membrane. The osmotic pressure of a fluid is the sum of the partial pressure contributed by each of the solutes in that fluid, the effective osmotic pressure is dependent on those substances that fail to pass through the pores of the semipermeable membrane. Sodium which is the principal cation of the extra cellular fluid, countributes a major portion to the osmotic pressure (90%). Since the cell membranes are completely permeable to water, the effective osmotic pressures in the two compartments are considered to be equal. Any condition that alters the effective osmotic pressure in either compartment will result in redistribution of water between the compartments. Thus an increase in effective osmotic pressure in the extra cellular fluid, which would occur most frequently as a result of increased sodium concentration will cause a net transfer of water and would continue until the effective osmotic pressure in the two compartments were equal. Thus the intracellular fluid shares in losses that involve a change in concentration or composition of the extra cellular fluid but shares slowly in changes involving loss of isotonic volume alone.

Serum osmolality, normally 289±9 mmol/kg. measures the total concentration of all osmotically active entities in the serum water, like other measurements of concentration it does not of itself give information about the total amounts of circulating materials which depends on the plasma volume as well as the concentration. Additional information is obtained if urinary osmolality is measured simultaneously as this reflects the action of anti diuretic hormone on renal tubules.

Increase in serum osmolality is a consequence of either an increase in serum sodium concentration or in the concentration of other osmotically active substances.

The decrease in serum osmolality is almost always attributable to a low sodium concentration. Although this may be due to sodium deficiency the more marked falls are seen in conditions with water retention without sodium retention. Excess A.D.H. activity, which is a feature of the body's response to injury may also lead to a low serum osmolality particularly if excessive intravenous administration of isotonic glucose solution is carried out after trauma or in the immediate post operative period.

<u>Osmolar gap</u> - The difference between the measured plasma osmolarity and osmolarity predicted from the measured (Na^+) is termed the osmolar gap.

EFFECT OF ANAESTHESIA ON THE SODIUM BALANCE

Fall in serum sodium level is well known to occur after any surgery (Fleer et al 1980 and Chan et al 1980). Changes in fluid and electrolyte metabolism as a result of trauma are for the most part the result of alterations in the systemic neuroendocrinal environment. In order for a reflex to be initiated, the stimulus must be perceived by a specialized receptor that transduces the stimulus in to electrical activity and transmits it to the brain. This is exemplified by the experiments of Hume and Egdah (1959), in which one hindlimb of a dog was left attached to the body only by the femoral nerve, artery and vein. The trauma to the innervated but other wise detached hind limb continued to evoke an increased A.C.T.H. and cortisol response. When the nerve was severed, leaving only the artery and vein intact the response to trauma was eliminated. Similarly patients under going lower limb surgery under spinal anaesthesia do not demonstrate an increase in vasopressin secretion during the procedure as compared to patients under going the same procedure under general anaesthesia. This is because of the inhibiting effect of spinal anaesthesia on the neural pathways. Laparotomies in absence of diminished circulatory volume do not result in adenocortical stimulation, if the traumatized area is denervated. Similarly,local anaesthetics by blocking the transmission of efferent impulses from the area of injury, inhibit the neuroendocrinal response to

operative trauma elecited by stimuli present at the operative site. The perception of stimulus need not be conscious, as evidenced by the ability of individuals to respond to surgical stimuli despite the presence of general anaesthesia. Even this response may not be the same had anaesthesia not been present. This difference arises at least in part, through the ability of general anaesthetics them self to intiate, inhibit or augment neuroendocrinal reflexes. No operative trauma ought to be thought of with out a consideration of the particular anaesthetic agent employed and depth and duration of anaesthesia.

THE SODIUM RESPONSE TO SURGICAL TRAUMA

Any surgical trauma causes a sudden rise in aldosterone and cortisol level in the patient (Liaurado J.C. and N.F.Woodruff 1957). The aldosterone and cortisol, both are responsible for sodium retention in the post operative period (Jepson R.P., K.M.Endocr 1951). The decreased renal excretion ofsodium is a well documented feature of the post operative period (Hardey J.D. and I.S.Ravdin 1952). Functional extra cellular fluid volume has recently been shown to be a major determinent of the magnitude of renal sodium excretion in the normal individual. (Epstein F. H., 1957). The reduction of functional extra cellular fluid volume during the operative procedure is independent of whole blood loss during operation. The only factor observed which would tend to influence the degree of functional extra cellular

loss is the magnitude of the local trauma. Thus a decrease in functional extra cellular fluid volume during the post operative period is due to an internal redistribution of fluid. This decrease in functional extra cellular volume in it self is a strong stimulus for aldosterone secretion despite an overall fluid retention. This reduction of ECF volume in turn is responsible for sodium retention in the post operative period. Thus normally there is sodium retention in post operative patients.

A fall in plasma sodium concentration often to hyponatraemic level, is well known to occur after trauma and major surgery (Flear CTG, Bhattacharya S.S., Singh C.M. 1971 Chan S. Radcliffe & Johnson A 1980). It is widely believed that the fall in plasma sodium after uncomplicated surgery results from exogenous dilution, but this is often insufficient to cause all of the observed changes (Flear CTG, Bhattacharya S.S., Singh C.M. 1971). In patients severely ill after operation profound fall in plasma sodium may occur and osmolar gaps are seen (Tindall S.F., Clark R.G., 1976 and Flear CTG, Singh C.M. 1963). The lowering of plasma sodium may be abrupt or slow and mustained. Abrupt fall in sodium plasma are often accompanied by osmolar gap that are both dynamic and changing (Flear CTG, Singh C.M. 1973). Sustained fall in plasma sodium are accompanied by reduced osmolality. The sick cell concept attributes osmolar gaps to isomotic redistribution of solute, from cells to extra cellular fluid, caused by an abrupt increase in cell

membrane premiability; and the sustained dilution with no osmolar gaps to a wide spread impaired capability of cells to maintain their normal content of non diffusible solute. (Flear C.T.G. 1970, Flear C.T.G., Singh C.M. 1973 and Flear C.T.G., Singh C.M. 1972).

The fall in serum sodium has been seen in the presence of sodium retention after trauma, inspite of a raised aldosterone level. Part of this hyponatraemia can be explanable on the basis of an obligatory antidiureases due to raised anti diuretic hormone level lasting for 24-36 hours (Le Quesne and Lewis 1952) post surgery.

The plasma aldosterone concentration demonstrates a circadian rhythm in which the peak concentration occurs at mid morning and the lowest concentration in late afternoon and night. Following trauma, surgery, the circadian rhythm is lost and elevated concentrations are observed during the entire 24 hours period. Plasma concentration of aldosteronone also increased following anaesthesia alone, but not to the extent seen, following injury and major operations. The highest concentration of aldosterone has been noted in the agonal period following injury.

Cells of the adrenal cortex zona glomerulosa synthesize and secrete aldosterone in response to stimuli. Following surgery, the two most important mechanisms for aldosterone secretion appears to be through A.C.T.H. and

angiotensin, stress induced elevations in aldosterone are probably mediated through A.C.T.H. The stimulatory effect of A.C.T.H. on aldosterone production is short lived. As a result of this short lived potency, A.C.T.H. probably has a minor role in chronic states where Angiotensin II appears to be the main stimulating hormone, which in addition also has a stimulating role even in the early phase of injury. Other factors that may alter the aldosterone secretion by the adrenal cortex are -

a. Increased P_K (Plasma potassium)
b. Decreased P_{Na} (Plasma sodium)

Increase in PK represent an important stimuli for aldosterone secretion, but do not represent a mechanisms for changing aldosterone secretion when sodium in take changes. The increased aldosterone secretion seen with decreased plasma sodium represent an appropriate response for maintaining sodium balance. However, the effect of plasma sodium on aldosterone secretion is of minor importance in the regulation of sodium excretion for two reasons. First of all, decrease in plasma sodium have a relatively weak stimulatory effect on aldosterone secretion secondly changes in sodium intake have minimal effects on plasma sodium. For example, while an increased sodium intake adds sodium to the extra cellular fluid and produces a transient increase in plasma sodium the plasma osmolality also

increases, stimulating the osmoreceptors. The resulting stimulation of thirst and ADH. release leads to expansion of the plasma volume and dilution of the ingested sodium, so that the overall changes in plasma sodium is small. Thus, the changes in aldosterone secretion that accompany changes in sodium intake must be primarily mediated by Angiotensin II.

Primary action of aldosterone is related to fluid and electrolyte balance. In the early distal convoluted tubule, Aldosterone increases the reabsorption of sodium and of chloride and in the late convoluted tubule and in early collecting duct it promotes the reabsorption of sodium and the excretion of potassium. Thus the aldosterone level during and after surgery (trauma), is responsible for the sodium retention, but hyponatremia in post operative period is believed to be provoked by an even greater gain of water (C.M.Singh and C.T.G. Flear 1968) due to persistent elevated level of anti-diuretic hormone in serum. In experimental animals when the factors that regulate sodium excretion like G.F.R. and aldosterone, are controlled, an animal can still regulate sodium excretion to match sodium input. For example, in an experimental animal in whome a constant G.F.R. is maintained by controlling blood flow to the kidneys and a high plasma concentration of aldosterone is maintained by administering large doses of the hormone, intravenous infusion of isotonic

saline still will be followed by a decrease in sodium reabsorption and hence an increase in sodium excretion. The phenomenon whereby an increase in sodium input can result in an increase in sodium excretion independent of any significant increase in G.F.R. or decrease in aldosterone level is termed the 'THIRD FACTOR EFFECT'. Conversely, a decrease in sodium input can result in a decrease in sodium excretion independent of any significant change in G.F.R. or aldosterone level, a phenomenon that can be referred to as the absence of third factor.

Despite intensive investigations, the mechanism for the third factor effect remains poorly understood (De Wardener 1978). The third factor effect probably involves several different mechanisms because changes in sodium reabsorption in both the proximal tubule and distal nephron are observed. With small increase in sodium intake, the third factor effect appears to be primarily due to a decrease in sodium reabsorption in the medullary collecting duct. It has been postulated that this decrease in sodium reabsorption is mediated by prostaglandins, bradykinin, or an as yet unidentified" natriuretic" hormone. When large quantities of sodium are administered (e.g. by intravenous infusion). Sodium reabsorption in the proximal tubule is depressed correspondings. This decrease in proximal tubular sodium reabsorption can be attributed at least in part to the dependence of proximal tubular water and solute reabsorption on the hydrostatic and oncotic pressures in the peritubule

capillaries. According to the starling principle, the rate of fluid movement, from capillaries to interstitial space (i.e. the rate of filteration) is proportional to the difference between the hydrostatic and oncotic pressure gradients across the capillary wall, the so called net filtration pressure.

Rate of filtration is directly proportional to $(Pc - P_i) - (\pi c - \pi_i)$. The rate of fluid movement from interstitial space to capillaries (ie. the rate of reabsorption) is therefore proportional to $(P_i - Pc) - (\pi i - \pi c)$.

if the above equation is applied to the reabsorption of fluid from the peritubular interstitial space into the peritubular capillaries, it becomes evident that an increase in peritubular capillary hydrostatic pressure (pc) or a decrease in peritubular capillary oncotic pressure (πc) will retard the reabsorption of fluid into the capillaries. The movement of fluid from the lateral intracellular space to the peritubular space will therefore be retarded and the hydrostatic pressure in the lateral space would increase. This increased hydrostatic pressure, in turn, will impair the reabsorption of water and solutes by the proximal tubule, perhaps by allowing water and solutes that already have been transported into the lateral space to leak back (pump leak) in to the tubular lumen. Ingestion of a large quantity of sodium could increase peritubular capillary hydrostatic

pressure and decrease peritubular capillary oncotic pressure, there by decreasing the reabsorption of sodium by the proximal tubule, such change in peritubular capillary hydrostatic and oncotic pressure also would decreases the reabsorption of water and the other solutes by the proximal tubules, thus accounting for the observation that all proximal tubular reabsorption is decreased following the ingestion of a large quantities of sodium or as a result of plasma volume expansion due to other causes. It should be noted that while the changes in hydrostatic and oncotic pressure could account for the decrease in proximal tubular sodium reabsorption following a large increase in sodium intake, many investigators believe that a hormone also may be involved. It is not known whether this 'natriuretic hormone' is the same as the hormone postulated to decrease sodium reabsorption in the medullary collecting duct in response to small increase in sodium. Although thedetailed mechanism for the third factor effect are not completely understood the participation of both the proximal tubule and medullary collecting duct occur in a logical manner with small increase in sodium intake, the third factor effect occurs in a region of the nephron that reabsorbs small quantities of sodium and "fine tunes" the rate of sodium excretion i.e. the medullary collecting duct. With large increases in sodium intake, thethird factor effect also occurs in the region that reabsorbs the large quantity of sodium i.e. the proximal tubule.

The other hormone playing an important role in the sodium and water regulation is, Arginine vasopressin (antidiuretic hormone), which is the primary hormone of the neurohypophysis in human beings. This hormone is synthesized in the hypothalamus and then transported to the neurohypophysis, where it is stored, until neural, signals to the neurohypophysis stimulate its release. Arginine vasopressin synthesis takes place in cells of the supraoptic and para venticular nuclei located in the anterior hypothalamus. Various stimuli are responsible for the alteration in secretion of Arginine vasopressin, most of which arise to a great some extent as a result of trauma. Therefore it is no surprise that the secretion of vasopressin is increased after a major surgical trauma.

Moran et al (1964) have identified four phases of vasopressin secretion following surgery. The first phase is the normal preoperative control period in which plasma vasopressin concentration is within the normal range. The second phase consists of a mild elevation that results from the over night fast. This period can be abolished by the administration of intravenous fluids during the pre operative period. The third phase results from cutaneous & visceral stimuli and lasts from skin incision to closure. This phase is characterized by transient elevation of antidiuretic hormone (vasopressin) concentration that returns to normal value. The fourth phase corresponds to thepost operative phase in which there is an early increase in the plasma vasopressin concentration followed by a return to normal

value by the fifth post operative day.

Moran et al (1964) also hypothetized that there are four efferent reflexes controlling the vasopressin release and each of these can over ride the preceding one three of these reflexes. Osmoreceptor, baroreceptor and left atrial strech receptor reflex are negative feed loops. The fourth reflex is thought to be mediated through painfuls stimuli and is not a feed back loop. Therefore in the presence of pain vasopressin secretion can occur in the face of a hypoosmolar, hypovolumic condition that would normally inhibit vasopressin secretion and may explain the persistent elevation of vasopressin secretion seen for 5-7 days following surgery. The persistant secretion of vasopressin produces a low urinary output with high osmolality and profound dilutional hyponatraemia.

Any surgery, produces rapid changes in functional extracellular fluid volume (Shires et al 1961), effective circulatory volume, extracellular osmolality and electrolyte composition, that results in the stimulation of the neuroendocrine system. Thus the neuroendocrinal response induces alteration in the renal and circulatory functions which can then alter the salt and water balance as required.

The increase in plasma vasopressin lasts for 3-5 days after surgery under and in most circumstances it results in water retention and oliguria. Post operative

oliguria was originally believed to be a normal accompaniment of surgery that did not particularly have any ill effects. Although oliguria is well tolerated in most forms of mild to moderate surgery, it may be potentially harmfull.

Shires et al (1961) hypothetized that a significant loss of the fluid in 3rd space may account in part for the hyponatraemia. The osmolar gaps seen in the post operative patients could have been due to an isoosmolar redistribution of solute caused by an increased cell membrane permeability (Flear and Singh, 1983, Flear and Singh 1978, and Flear and Singh 1982).

ROLE OF FLUID ADMINISTRATION IN SURGICAL PATIENTS

That dilution plays an important part in post operative hyponatraemia is unanimously accepted (Chan et al 1980). Thus serum sodium level can be effected by the type of fluid, which is given, in two ways. The first is that it predisposes to acute tubular necrosis in patients with severe trauma in whom hypovolumia and hypotension are apt to occur, and the second is that it sets the stage for the development of water intoxication (severe dilutional hyponatraemia, if large volumes of solute free fluids are given to the patient before, during or immediately after the operative event. Thus the most common electrolyte abnormality seen following surgery namely hyponatraemia, is partly as a result of the administration of hypotonic fluid even under conditions that favours salt and water retention.

The action of vasopressin in effecting water retention requires the presence of an intact counter current mechanism in the loop of Henle. This counter current mechanism is disrupted by a fall in medullary osmolality since the maintenance of normal medullary osmotic gradient requires the adequate delivery of sodium and chloride, to the long loops of Henle, which is decreased frequently after injury. The action of vasopressin is then impaired resulting in a defect in the urinary concentrating ability. Thus abnormal or increased urine output in a hypotensive or surgical patients does not reflect an adequate blood volume. In order to combat the fall in the medullary gradient following surgery, adequate tubular fluid flow must be ensured and maximal sodium reabsorption in the proximal nephron must be avoided. This is accomplished by the administration ofliberal amounts of salt solution such as ringer lactate or normal saline in the early post operative period.

The administration of normal saline in the early post operative per.od may result in a marked positive sodium and volute balance which may cause oedema. During this period of increased vasopressin secretion the urine volume can not be increased by the administration of water alone. It is the solute load that determines the urine volume and free water clearance during this period. An increase of urine output will occur only after the extracellular fluid space has been expanded by increasing the solute load. This increased urine output may result in a puffy patient post operatively, but

maximizes the protection of renal function.

That dilution alone is insufficient to account for the fall observed in the serum sodium, has been shown by the relationship between the serum osmolality and the serum sodium (Flear et al 1980 and Singh and Singh, 1971), in the peroperative and immediate post operative period. Among the two phases of water retention after surgery described by LeQuesne & Lewis (1953), the initial obligatory antidiuretic phase occured irrespective of sodium content of the fluid infused and was not suppressed by a strong hypotonic stimuli of dilutional hyponatremia. Which occured if the patient is given only dextrose solution or by the isotonic expansion of extra cellular space if patient is given isotonic solution. This could be because the water retention which is taking place, is controlled by vasopressin (Thomas and Morgan 1979 and Sinnetamby et al 1974) and sodium retention by aldosterone and other factors (Cochren 1978). The second phase of fluid retention which lasts from 36-120 hours is effected by the sodium contentof infused fluid. Therefore administration ofsodium free fluid leads to hyponatraemia with diuresis with free water loss (Tindall & Clark 1981). This may be either because of the sodium wash out effect in diuresis or the resetting of osmoreceptor in hypothalamus on a lower level (Robertson and Ather 1976). The patients who has been given only saline on the other hand tends to retain water and maintain the plasma sodium

level. The reason for sodium retention and the normal serum sodium level is difficult to understood. There may be several explanations forthe o-ver riding of the vasopressin response in thepresence of isotonic expansion of the extracellular compartment. The gradual expansion of the extracellular space may have allowed stretch receptors in the capacitance vessels to readjust with out increasing the vasopressin secretion. Alternatively the kidney may have developed a reduced ability to excrete sodium in the late post operative period and this could have led to sodium retention in the presence of a high sodium in take (Ruidal et al 1981).

In post operative period there is mild to moderate hyponatraemia with hyperkalemia this is primarily brought about by the secretion of vasopressin. Plus the one hydration of the patients with non solute containing fluid. The potassium level may be some what elevated because potassium is lost from the cells as a consequence of surgical trauma and corticosteroid level and starvation.

Patients after surgery have a diminished urinary excretion of sodium and they do not excrete all the sodium load this sodium retention has been attributed to a reduced plasma volume. Fleer and Clark (1955) observed that sodium retention did not occur after trauma. If the patient were given adequate blood transfusion or isotonic solution. Irvin et al (1972) reported that the urine sodium

did not fall in patients after surgery if they were given balanced salt solution during as well as after the surgery.

The fall in plasma sodium which occurs in the majority of patients given dextrose or dextrose saline after surgery is much smaller than seen that in patients with symptomatic water intoxication after surgery (Dentsen et al 1966).

MATERIAL AND METHOD

MATERIAL AND METHOD

The study of serum electrolytes was done on three groups of patients, 15 to 60 years under going surgery and requiring fluid infusion for at least 24 hours post operatively.

The patients were divided into 3 groups according to nature of fluid infused in post operative period.

Group I - 3 Lt. of 5% dextrose/day.

Group II - 1 Lt. of isotonic saline + 2 Lt. 5% dextrose.

Group III - 2 Lt. of isotonic saline + 1 Lt. 5% dextrose.

Investigation done

1. Measurement of B.P. in the - lying down posture and
   - sitting posture
2. Haematocrit.
3. Serum studies
   a. Serum sodium
   b. Serum potassium
   c. Serum osmolality
   d. Blood sugar level
   e. Blood urea level
4. Urine analysis
   a. Volume / 24 hours
   b. Urine sodium excretion / 24 hours
   c. Specific gravity

5. Body weight

On the day before operation patient were starved from mid night except in emergency surgery. The intravenous fluid was administered through peripheral veins. All the patients received the same drug during anaesthesia and adequate identical analgesia after operation. The blood samples were taken at 0900 hour from peripheral vein on - 1 (One day prior of operation, '0' Immediate post operative), + 1 (First post operative + 2 (Second post operative day), by the standard technique with a sterilized syringe and needle.

24 hour urine was collected in a measuring flask or via a catheter to measure the exact 24 hour urine volume.

The serum sodium, potassium and urinary sodium were measured by flame photometer (systonic ahmedabad). in the department of Biochemistry, M.L.B. Medical College, Jhansi.

Principle

When small quantity of metal salts such as of sodium or potassium is introduced into a flame, a characteristic light is emitted. The measurement of the intensity of such emission and its correlation with the concentration of the element is the basis of flame photometry.

The instrument made on this principle is called flame photometer. The flame photometer has the following parts.

1. The air pressure regulator and flow meter for the fuelgas.
2. The atomizer
3. The Burner
4. Optical system
5. Photocells
6. Recorder - A galvanometer with light spot or needle.

For the fuel gas, coocking gas cylinder is convenient. Compressed air is used to atomize the sample and carry it to non luminous flame. Both gas and air supplies are carefully regulated to maintain constant flow rate of the samples into the flame.

The solution is sprayed as a fine mist of droplets in to the non luminous flame which becomes coloured by the characteristic emission of the metal light of wave lengths which corresponds to the element being determined is isolated by the use of a light filter or prism system and allow to fall on photocells. The electric current generated is measured. This is indicated by the light spot on the recorder.

<u>Determination of serum sodium and potassium & urinary sodium</u>

<u>Material & reagents</u>

1. Flame photometer
2. Gas cylinder

3. Polythene bottles (500 mg capacity) for standard solution.
4. Polythene container for distilled water.
5. Polythene small cuvettes for aspirating the test solutions in the flame.
6. Double distilled or deionized water
7. Polythene small tubes
8. Stock sodium standard (200 meq/lt.) dissolve 11.69 gm of pure dry sodium chloride (A.R.) in one littre of water.
9. Stock potassium standard (10 meq/lt.) dissolve .746 gm of pure dry potassium chloride (AR) in one littre of water.
10. Combined working standards of sodium and potassium.

Procedure

Sodium :- Put the light filter (580 -590 um yellow green) in the filter socket, adjust the gas adjusting knob gradually until the individual blue cones of the flame become separated. Then adjustment of glavanometer is done, first with distilled water & then with maximum strength working solution. Then aspirate one by one standard solution and note the glavanometer reading & then calculate sodium level.

Potassium :- The potassium light filter (766-770 mm, Red) The instrument is standerdzed and the same test solution is aspirated & the reading is noted and potassium value calculated.

Urinary Sodium :- Dilute the urine 1 to 100 ml and measurement is done as for blood sodium.

It is some times helpful to calculate the osmolality from the molar concentrations of the main osmotically active subtances. For both serum and urine this can be done if the molar concentrations of sodium (Na^+) potassium K^+, urea and glucose are known. The serum osmolality is calculated by the formula (Harrison's principles of internal medicine - 2 elevinth edition, Page No. 1791).

Serum osmolality (mosmol/lt.)

$$+ = 2\,(Na^+) + (K^+) + \frac{\text{Glucose/mg/dl}}{18} + \frac{\text{BUN (mg/dl)}}{2.8}$$

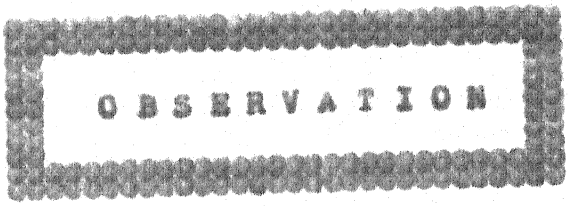
For most normal sera this is close to 2 (Na^++K) and for normal urine, glucose can be ignored. Comparison of calculated osmolality with that actually determined is often helpful in pointing to the presence of some previously unsuspected osmotically active substance.

The specific gravity of urine was measure by urometer.

The weight of patient was recorded at 0900 hours each day after correcting it for the weight loss resulting from the removal of the surgical specimen.

Intravenous fluid given

All the groups of patients redeived identical volumes of fluid in early post operative period i.e. 3 lt./ day. But nature of fluid was according to the groups already mentioned.


OBSERVATION

O B S E R V A T I O N

The present study was done in our institute, M.L.B. Medical College, Hospital, Jhansi between July 1988 and July, 1989. During this period we studied the effect of perioperative fluid infusion on serum electrolytes in 168 patients who under went various types of surgery.

The patients were divided in three groups according the type of fluid infused in peri-operative period.

Out of 168 patients, 60 patients received 3 lt. 5% dextrose/day, 60 received 2 lt. 5% dextrose and 1 lt. normal saline/day and the rest 48 patients received 1 lt. 5% dextrose and 2 lt. normal saline/day.

All 168 patients were studied for serum electrolytes (sodium and potassium), serum osmolality, urine volume and urinary sodium.

All the tests were done by one person under identical conditions.

SERUM SODIUM (TABLE I)

- Among the three groups, patients receiving 3 lt. of 5% dextrose showed a significant fall (P/ .001) in serum sodium on first post operative day which persisted on the second post operative day.

- Patients receiving 2 lt. of 5% dextrose + 1 lt. normal saline showed a significant fall (P$<$.05) in serum sodium on the first post operative day which showed a further progression (P$<$.001) on the second post operative day also.
- Patient receiving 1 lt. 5% dextrose + 2 lt. normal saline showed a significant rise (P $<$.001) in serum sodium on both the first and 2nd post operative days.

SERUM POTASSIUM (TABLE II)

- Patients receiving 3 lt. of 5% dextrose/day showed a significant rise (P $<$.05) on the first post operative day and an insignificant rise (P $<$.1) on the second day.
- Patients receiving 2 lt. 5% dextrose + 1 lt. normal saline/day showed a significant rise (P$<$.001) persisting upto the second post operative day.
- Patients receiving 1 lt. 5% dextrose + 2 lt. normal saline showed a significant rise (P$<$.001) on first post operative day which persisted on second day.

SERUM OSMOLALITY (TABLE III)

- Patients receiving 3 lt. 5% dextrose/day showed a significant fall (P$<$.001) in serum osmolality on first post operative day which persisted on second day.
- Patients receiving 2 lt. 5% dextrose + 1 lt. normal saline showed a significant fall (P$<$.02) on first post operative day which progressed (P$<$.001) on second day.

- Patients receiving 1 lt. 5% dextrose + 2 lt. normal saline showed no significant (P/.5) change in serum osmolality on first and second post operative day.

URINE OUTPUT (TABLE IV)

- All the three groups of patients receiving 3 lt. of 5% dextrose, 2 lt. 5% dextrose + 1 lt. normal saline/day respectively, showed a significant rise (P/.001) in urine output on first post operative day which persisted on second day.

URINARY SODIUM (TABLE V)

- Patient groups receiving 3 lt. 5% dextrose and 2 lt. 5% dextrose + 1 lt. normal saline respectively showed a significant fall in urinary sodium excretion on first and second (P/.001) post operative day.
- Patients receiving 1 lt. 5% dextrose + 2 lt. normal saline showed an insignificant rise (P/.5) on first post operative day but progressed to a significant level (P/.001) on secondp post operative day.

INCIDENCE OF HYPONATRAEMIA IN VARIOUS GROUPS (TABLE VI)

- In group I (i.e. patient received 3 lt. 5% dextrose). 10% patients were hyponatraemic on preoperative day but after receiving 3 lt. 5% dextrose 60% patients became hyponatraemic on first post operative day.
- In group II (patient receiving 2 lt. 5% dextrose + 1 lt. normal saline).

10% patients were hypnatraemic on pre operative day but after receiving 2 lt. 5% dextrose + 1 lt. normal saline. 30% patients became hyponatraemic on first post operative day.

- In group III (patients receiving 1 lt. 5% dextrose + 2 lt. normal saline per day) none of the patients were hyponatraemic on first or second post operative day.

TABLE NO. I

Mean values of serum sodium in patients under going surgical procedure.

Sl. No.	Nature of fluid infused	No. of patients	Values	Pr. O. mmol/lt.	Po. O. day 1 m mol/lt.	Po. O. day 2 m mol/lt.
1.	3 Lt. 5% Dextrose	60	Mean	144.2	136.4	135.8
			S.D.	±4.83	±5.35	±5.95
			P	-	< .001	< .001
2.	2 Lt. 5% Dextrose + 1 lt. normal saline	60	Mean	141.7	139.5	137.3
			S.D.	±4.57	±5.26	±5.56
			P	-	<.05	< .001
3.	1 Lt. 5% Dextrose + 2 lt. normal saline	48	Mean	140.75	142.00	142.2
			S.D.	±3.95	±2.33	±2.66
			P	-	<.001	<.001

Pr.O = Pre-operative

Po.O = Post-operative

S.D. = Standerd Deviation

'p' value - Po 1 and Po 2 were calculated in relation to Pr.O.

TABLE NO. II

Mean values of serum potassium in patients under going surgical procedure.

Sl. No.	Nature of fluid infused	No. of patients	Values	Pr. O. m.mol/lt.	Po. O. day 1 m.mol/lt.	Po. O. day 2 m.mol/lt.
1.	3 Lt. 5% Dextrose	60	Mean	3.87	3.91	3.80
			S.D.	±.24	±.04	±.32
			P	-	∠.05	∠.1
2.	2 Lt. 5% Dextrose + 1 lt. normal saline	60	Mean	3.77	4.08	4.08
			S.D.	±.72	±.38	±.55
			P	-	∠.001	∠.001
3.	1 Lt. 5% Dextrose + 2 lt. normal saline	48	Mean	3.15	3.83	3.78
			S.D.	+_.43	±.43	±.42
			P	-	∠.001	∠.001

Pr.O. = Pre-operative

Po.O. = Post-operative

S.D. = Standard Deviation

'p' value - Po.O 1 and Po.O 2 were calculated in relation to Pr.O.

TABLE NO. III

Mean value of serum osmolality in patients under going surgical procedure.

Sl. No.	Nature of fluid infused (24 hours)	No. of patients	Values	Pr.O. m.mol/lt.	Po. O. day-1 m.mol/lt.	Po. O. day-2 m.mol/lt.
1.	3 Lt. 5% Dextrose	60	Mean	301.36	286.54	284.37
			S.D.	±12.07	±11.81	±12.12
			P	-	∠.001	∠.001
2.	2 Lt. 5% Dextrose + 1 lt. normal saline	60	Mean	292.59	289.09	286.69
			S.D.	±12.46	±19.70	±14.84
			P	-	∠.02	∠.001
3.	1 Lt. 5% Dextrose + 2 lt. normal saline	48	Mean	296.53	296.26	296.69
			S.D.	±6.35	± 7.29	± 5.79
			P	-	∠.5	∠.5

Pr. O. = Pre-operative

Po. O. = Post-operative

S.D. = Standard Deviation

'p' value - Po. O.1 and Po.O. 2 were calculated in relation to Pr.O.

TABLE NO. IV

Mean urine output values in patients under going surgical procedure.

Sl. No.	Nature of fluid infused (24 hours)	No. of patients	Value	Pr. O. ml/24hrs.	Po. O. day-1 ml/24hrs.	Po. O. day-2 ml/24hrs.
1.	3 Lt. 5% Dextrose	60	Mean	1140	2610	3115
			S.D.	±118.32	±363.69	±649.17
			P	-	< .001	< .001
2.	2 Lt. 5% Dextrose + 1 lt. normal saline	60	Mean	1075	1840	2440
			S.D.	±133.87	±88.06	±176.76
			P	-	< .001	< .001
3.	1 Lt. 5% Dextrose + 2 lt. normal saline	48	Mean	1012.5	1406.25	1687.5
			S.D.	±106.40	±106.44	±138.37
			P	-	< .001	< .001

Pr. O. = Pre-operative

Po. O. = Post-operative

S.D. = Standard Deviation

'p' value - Po.O.1 and Po.O. 2 were calculated in relation to Pr.O.

TABLE NO. V

Mean value of urinary sodium excretion / 24 hours in patients under going surgical procedure.

Sl. No.	Nature of fluid infused	No. of patients	Values	Pr. O. meq/24hrs.	Po. O. day-1 meq/24hrs.	Po. O. day-2 meq/24hrs.
1.	3 Lt. 5% Dextrose	60	Mean	116	45.7	43.6
			S.D.	±26.74	± 18.31	±33.24
			P	-	∠.001	∠.001
2.	2 Lt. 5% Dextrose + 1 lt. normal saline	60	Mean	140.5	76	71.6
			S.D.	±19.88	± 17.42	±17.76
			P	-	∠.001	∠.001
3.	1 Lt. 5% Dextrose + 2 lt. normal saline	48	Mean	133	133	140.12
			S.D.	±24.46	± 7.94	±16.26
			P	-	∠.5	∠.001

Pr. O. = Pre-operative

Po. O. = Post-operative

S.D. = Standard Deviation

'p' value = Po.O. 1 and Po.O. 2 were calculated in relation to Pr.O.

TABLE NO. VI

Incidence of hyponatraemia (P Na ⩽ 137 meq/lt.) on pre operative and first post operative day.

Sl. No.	Nature of fluid infused (24 hours)	No. of patients	Pr. O. Day	Po. O Day-1
1.	3 Lt. 5% Dextrose	60	6 (10%)	36 (60%)
2.	2 Lt. 5% Dextrose + 1 lt. normal saline	60	6 (10%)	18 (30%)
3.	1 Lt. 5% Dextrose + 1 lt. normal saline	48	12 (25%)	0 (0%)

Pr.O. = Pre-operative

Po.O. = Post-operative

DISCUSSION

D I S C U S S I O N

SERUM SODIUM IN RELATION TO SURGICAL PROCEDURE AND FLUID ADMINISTRATION

The serum sodium level normally represents the degree of dilution or concentration of body fluid both in health and disease. Retention of sodium, reduction in urinary sodium and absence of free water excretion are the classic responses to surgical trauma. The surgical trauma causes a sudden rise in aldosterone and cortisol level. The cortisol and aldosterone both are responsible for the sodium retention and reduction in urinary sodium in post operative period (Jepson R.P., K.M. Chadon, 1951). Following surgery, important mechanism for aldosterone secretion appears to be through A.C.T.H. and angiotensin, stress induced elevation in aldosterone are probably mediated through A.C.T.H. The stimulatory effect of A.C.T.H. on aldosterone production is short lived. As a result of this short lived potency, A.C.T.H. probably has a minor role in chronic states where angiotensin II appears to be the main stimulatory hormone, which in addition also has a stimulatory role even in the early phase of injury.

Other factors that may alter the aldosterone secretion by the adrenal cortex are -

1. Increased PK (Plasma potassium)

2. Decreased P Na (Plasma sodium)

Increase in plasma potassium represent an important stimulus for aldosterone secretion, but donot represent a mechanism for changing aldosterone secretion, when sodium intake changes. The increased aldosterone secretion seen with decreased plasma sodium represents an appropriate response for maintaining sodium blance.

However the effect of plasma sodium on aldosterone secretion is of minor importance in the regulation of sodium excretion for two reasons first of all, decrease in plasma sodium have a relatively weak stimulatory effect on aldosterone secretion, secondarely changes in sodium intake have minimal effect on plasma sodium for example, while an increased sodium intake, add sodium to the extra cellular fluid and produces a transient increase in plasma sodium, the plasma osmolality also increases, stimulating the osmoreceptor. The resulting stimulations of thirst and A.D.H. release leads to expansion of the plasma volume and dilution of the ingested sodium. So that the over all change in plasma sodium is small. Thus the changes in aldosterone secretion that accompany changes in sodium intake must be primarily mediated by angiotensin II. The decreased renal excretion of sodium is well documented feature of the post operative period (Hardey, J.D. and I.S. Ravidin, 1952). The functional extra cellular fluid volume has recently been shown to be a another major determinant

of renal sodium excretion in the normal individual (Spatein F.H., 1957). The decrease in functional extra cellular fluid volume during the per operative period it self is a strong stimulus for aldosterone secretion, which causes sodium retention in the post operative period.

A fall in the plasma sodium concentration often to hyponatraemic levels is well known to occur even after trauma and surgical procedures (Flear C.T.G. Bhattacharya S.S., Singh C.N. (1971) and Chan S., Radcliffe A. Johnson A., 1980) inspite of an raised aldosterone level. Part of this hyponatraemia can be explainable on the basis of an obligatory antidiuresis due to a raised antidiuretic hormone level lasting for 24-36 hours (Le Quesne and Lewis, 1952 and Chan et al, 1980) post surgery. This hyponatraemia in post operative period is beleived to be provoked by an even greater gain of water (C.N. Singh and C.T.G. Flear, 1968). Another basis of post operative hyponatraemia was hypothetized by Shires et al (1961) who showed a significant loss of fluid with in the third space accounting in part for the hyponatraemia.

An other determinent of the serum sodium level is the type of fluid which is infused in the post operative period. If a large volume of solute free fluid is given to the patient in the peroperative period the most common electrolyte abnormality seen following surgery

is hyponatraemia. The raised aldosterone and cortisol level after surgery was the basis of the present concept of giving salt free fluids in theearly post operative period. During the planning of fluid therapy what had not been considered was the raised level of A.D.H., decreased renal excretion of sodium and loss of extra cellular fluid volume due to losses in the third space and dilutional hyponatraemia post surgically.

It is the balance between the sodium retaining factors and sodium dilution factors which ultimately determines the serum sodium level in thepost operative period. Thus the fluid administration in the perioperative period is of critical importance.

Our study clearly showed the importance of fluid administration on the serum sodium level. Other factors being identical only variable in our study was the amount of sodium administration in the post operative period. Thus the significant fall in serum sodium level persisting upto 48 hours post operative in patients receiving 3 lt. of pure 5% dextrose and 2 lt. 5% dextrose + 1 lt. normal saline, and a significant rise in patients receiving 2 lt. normal saline + 1 lt. 5% dextrose clearly bear out this fact what's of added importance is that patients with 3 lt. 5% dextrose had serum sodium level which can be classified as hyponatraemic (P Na / 137 meq/lt)

Thus our study shows similar results as shown by study of A.J.Guy, J.A. Michaels and T.T.G. Flear (1987) and flear C.T.G., Bhattacharya S.S. and Singh C.M. (1971) and Chan S., Redcliffe A. and Johnson A (1980). Their study showed that there is hyponatraemia in post operative period in patients undergoing surgical procedure and receiving salt free fluid in post operative period.

The study of A.J.Guy, J.A.Michaels and C.T.G. Flear, (1987) showed that about 27.5% patients, who underwent various types of surgical procedures became hyponatraemic on first post operative day, while our study shows that about 60% patients with 3 lt. 5% dextrose group and 30% patients with 2 lt. 5% dextrose and 1 lt. normal saline group became hyponatraemic. This difference is probably because our study is mainly based on patients who underwent major surgical procedure while their study comprises of patients undergoing minor, moderate and major surgical procedures.

In the present study, 48 patients who under went surgical procedure received 2 lt. normal saline + 1 lt. 5% dextrose per day. They never became hyponatraemic in post operative period, and no patient developed signs of hypernatraemia namely pulmonary or peripheral oedema. It can be stated that infusion of balanced salt solution to the patients under going surgical procedure prevents hyponatraemia and aids quick recovery of these patients

(A.J.Guy et al, 1987).

SERUM POTASSIUM IN RELATION TO SURGICAL PROCEDURE AND FLUID ADMINISTRATION

Serum potassium is usually found elevated after surgical procedures and elevation is usually dependent on the severity of surgical procedure. In cases of major surgery, elevation is more and with minor surgery elevation is less.

The present study shows a significant elevation in serum potassium on first and second post operative day in almost all the patients. This elevation is independent of nature of the post operative fluid infusion (i.e. 5% dextrose/normal saline). Our study differes from the study of A.J. Guy et al (1987). Their study showed a fall in serum potassium in post operative period. The fall was inversally proportional to severity of surgical procedures. But neither the nature of fluid administration nor the value of serum sodium have any effect on the serum K^+ level (A.G.Guy et al (1987). The reason for the elevated serum potassium level may be

1. Cell damage
2. Change in membrane potential
3. Alkalosis

All these three may occur after surgery which causes K^+ level, to move out of the cell leading to raised serum K^+ level.

SERUM OSMOLALITY IN RELATION TO SURGICAL PROCEDURE AND FLUID ADMINISTRATION

Sodium which is the principal cation of the extra cellular fluid contributes a major portion to the osmotic pressure and osmolality. The serum osmolality measures the total concentration of all osmotically active entities in the plasma water. Increase in serum osmolality is a consequence of either an increase in serum sodium concentration or in the concentration of other osmotically active substances. The decrease in serum osmolality is almost always attributable to a low serum sodium concentration. Although this may be due to sodium difficiency the more marked falls are seen in conditions with water retention, excess A.D.H. activity which is a feature of the body response to injury may also lead to a low serum osmolality particularly if excessive intrevenous administration of 5% dextrose is carried out after operation.

Present study shows a significant fall in serum osmolality in patients receiving 5% dextrose / 2 lt. of 5% dextrose + 1 lt. normal saline/day. This is probably because of dilutional hyponatraemia our findings agree with the study of T.T.Irvin., C.J. Hyter. V.K.Modgill. D.G. McDowell and J.C.Goligher (1972). They also observed a significant fall in serum osmolality in patients who

were kept on salt free fluid or dextrose saline solution. But serum osmolality also depends on blood urea nitrogen and blood glucose level. We did not find any significant change in either blood ureanitrogen nor in blood glucose level in any patients.

The present study shows no significant change in serum osmolality in patients of group III (Patients receiving 2 lt normal saline + 1 lt. 5% dextrose). The findings of a maintained serum osmolality in the presence of a significantly raised serum sodium do not agree with each other and this can only be explained on the basis of error or measurement. In the ultimate analysis we found a strong co relation between serum sodium and serum osmolality which indicates that dilution is the major factor in the hyponatraemia of uncomplicated surgical insults.

URINARY VOLUME IN RELATION TO SURGICAL PROCEDURE AND FLUID ADMINISTRATION

The present study shows almost similar preoperative urine volume in all groups of patients. The low urine output was seen on the day of operation in all groups (i.e. patients received 3 lt. 5% dextrose, 2 lt. 5% dextrose + 1 lt. normal saline and 1 lt. 5% dextrose + 2 lt. normal saline respectively. The patients receiving 3 lt. 5% dextrose per day showed a higher urine output in comparison to the patients receiving 1 lt. 5%

dextrose + 2 lt. normal saline on the first post operative day. The patients receiving 2 lt. normal saline + 1 lt. 5% dextrose showed a steady increase in urine output and urine output almost become similar on 3rd or 4th post operative day to the urine output of patients receiving 5% dextrose.

The present study shows a similar results as shown by study of S.F. Tindall, R.G.Clark (1981), and J.H.Thomas , D.B. Morgan (1979). J.H.Thomas et al (1979), observed that on the day of operation the arginine vasopressin (A.V.P.) increased in all groups of patients to the level which were much higher than those achieved by simple water depletion and much higher than would be expected from the plasma sodium concentration. On the subsequent days the urine, A.V.P. was higher in sodium group, where plasma sodium remained normal, than in the dextrose and dextrose + normal saline group, where there was a fall in the plasma sodium. These findings indicate that a suppression of A.V.P. secretion was not the mechanism that prevented the fall in plasma sodium concentration in saline group. They further suggested that a reduction in plasma volume or total extra cellular fluid volume, which would have been diminished or corrected by saline, was unlikely to be the cause of the increased A.V.P. in either group. The initial increase in A.V.P. in either group was therefore presumably a part of the stress response to the surgical operation (Moran et al, 1964).

vasopressin secretion seen for 5-7 days following surgery. The persistent secretion of vasopressin produces a low urinary output with high osmolality and profound dilutional hyponatraemia.

URINARY SODIUM IN RELATION TO SURGICAL PROCEDURE AND FLUID ADMINISTRATION

Our study shows a significant fall in urinary sodium in patients group receiving 3 lt. 5% dextrose/day and 2 lt. 5% dextrose + 1 lt. normal saline, while we did not find any significant change in urinary excretion in patients receiving = lt. 5% dextrose + 2 lt. normal saline. Results of our study for urinary sodium is similar to the findings of Tom Shires, Jack William M.D. , and Frank Brown M.D.(1961), and T.T. Irwin, V.K.Modgill, C.J.Hayter, Des. McDowell (1972).

We have calculated for our patients the minimum requirements to maintain water balance and to prevent the fall in plasma sodium. The calculation assumes that as the dextrose treated patients were hyponatraemic. They had no osmotic dirive to A.V.P. secretion (I.M.Thomas and D.B.Morgan, 1979). The fall in plasma sodium occured on day 1 and calculation suggests that in order to prevent this fall if 3 lt. of fluid were given which would have to contain at least 130 mmol/lt. of sodium chloride, to prevent the hyponatraemia.

Undoubtedly, the factors maintaining the fluid balance of the surgical patients are extremely complex and extra polations from the conclusions of studies of this type to more seriously ill patients must always be made with caution. Thomas and Morgan (1979) concluded from their studies that normal saline alone should be given during the early post operative period to avoid the development of hyponatraemia. The present study also shows that patients receiving 2 lt. normal saline + 1 lt. 5% dextrose resulted in the maintenance of plasma sodium at preoperative level, it did so at the expense of considerable salt and water retention which appeared to expand the extra cellular space, but not at the expense of causing clinical fluid overload.

When planning a fluid regimen it should not be forgoten that "third space lossess are not the true lossess, since the fluid and electrolyte envolved must eventually return to the normal pool of the extra cellular fluid unless they are lost through wound drainage Therefore as recovery progress the 'third space' contracts and an auto infusion taken place which may have to be taken in to account if the third space lossess were thought to be large initially.

The pathophysiology of the post operative water and salt balance remains a conjunctural field but a careful study of patients and the application of available knowledge should lead to improved patients care.

CONCLUSION

C O N C L U S I O N S

In the present study 168 patients were investigated to see the effect of the nature of peri-operative fluid infusion post operatively in surgical patients. We studied serum sodium, potassium, serum osmolality, urinary volume and urinary sodium excretion. Out of these 168 patients. 60 received 3 lt. 5% dextrose, other 60 received 2 lt. 5% dextrose + 1 lt. normal saline and rest 48 received 1 lt. 5% dextrose + 2 lt. normal saline.

The conclusions derived were as follows -

1. Serum sodium

The nature of fluid has a profound effect on the serum sodium levels, salt free solution i.e. 5% dextrose causes hyponatraemia in post operative period. while with balance salt solution hypernatraemia is seen in post operative period.

2. Serum potassium

The nature of fluid i.e. 5% dextrose or normal saline has no effect on serum potassium level. We found a rise in serum potassium in both group.

3. Serum osmolality

The nature of fluid has a significant effect on serum osmolality. Patients with 5% dextrose and 2 lt. 5% dextrose + 1 lt. normal saline showed a fall in

serum osmolality. While there was no change with 2 lt. normal saline + 1 lt. 5% dextrose.

4. Urine volume

Nature of fluid has an effect on the urine volume in the early post operative period, but on 3-4th day, urine volume become equal in all groups.

5. Urinary sodium

The salt free fluid (i.e. 5% dextrose) causes fall in urinary sodium excretion. While rise was seen in patients with 2 lt. normal saline + 1 lt. 5% dextrose.

Thus the present study shows that the infusion of salt free solution (i.e. 5% dextrose) in post operative period leads to hyponatraemia with falls in the serum osmolality and urinary sodium excretion. With the infusion of balance salt solution, there is no hyponatraemia and no fall in serum osmolality. Which in the long run improves the recovery of patients.

The exact cause of hyponatraemia and fall in serum osmolality can not be established by the present study which requires further work.

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SUMMARY

SUMMARY

In last three decades various reports had been published with a conclusion that salt free fluid infusion in the post operative period lead to hyponatraemia in surgical patients. The present work " An analysis of serum electrolytes and osmolality in surgical patients in reference to perioperative infusion" was carried out in the Department of Surgery and Biochemistry, M.L.B. Medical College, Jhansi, from July 1988 to July 1989, with the aim to assess the changes in serum electrolyte and osmolality in the patients, under going various surgical procedures & receiving different nature of fluid in perioperative period. The parameters studied were serum sodium, serum potassium, serum osmolality, blood urea nitrogen and blood glucose level, urinary volume and urinary sodium excretion.

During the study we investigated 168 patients for the above parameters, after deviding them into three groups, based on nature of fluid given in post operative period.

Group I. 60 patients receiving 3 lt. 5% dextrose/day.

Group II. 60 patients receiving 2 lt. 5% dextrose + 1 lt. normal saline.

Group III. 48 patients receiving 2 lt. normal saline + 1 lt. 5% dextrose.

From our observations we found

1. There was a significant fall in serum sodium in group I & II patients in post operative peried while in patients of group III a significant rise in serum sodium was found on first and second post operative day.
2. There was a significant rise in serum potassium level in the patients of all the three groups. This rise in serum potassium had no corelation with nature of fluid infused in the post operative period.
3. There was asignificant fall in serum osmolality in patients of group I & II but the fall was not observed in patients of group III.
4. The patients of all the three groups showed a significant rise in urine output on first post operative day which persisted on second day.
5. There was a significant fall in urinary sodium excretion in patients of group I & II, while the patients of group III. Showed a significant rise in urinary sodium excretion.
6. 10% patients were hyponatraemic in pre operative period in group I & II, but on first post operative day. 60% patients of group I and 30% of group II became hyponatraemic while in patients of group III, none of the patients were hyponatraemic on first post operative day.

The balance between the sodium retaining factors like raised aldosterone & cortisol, decrease renal urinary excretion and sodium dilutional factor like raised vasopressin, water retention and post operative salt free fluid administration, ultimately determine the, serum sodium level in post operative period. Thus the fluid administration in the peri-operative period is of critical importance. The present study clearly showed the effect of fluid administration on the serum sodium because the patients received salt free fluid become hyponatreamic in post operative period, while patients receiving 1 lt. 5% dextrose + 2 lt. normal saline their plasma sodium showed a significant rise of serum sodium level.

The pathophysiology of post operative water and salt balance is still not clear but the concensus of opinion agrees that the infusion of balance salts solution (according to our study 1 lt. 5% dextrose + 2 lt. normal saline) in early post operative period, prevents the hyponatraemia & improves the patient recovery.